Three had undergone successful DCR before the diagnosis of Giant fornix syndrome was made. Nine had corneal vascularisation and scarring before referral. Five had suffered spontaneous perforation or thinning. As in this case, all had deep upper fornices and associated changes of age-related dehiscence of the levator aponeurosis and universally, *S. aureus* was the inhabitant.

He postulated that gradual deepening of the upper fornix owing to age-related disinsertion of the levator aponeurosis allowed for the accumulation of a bacteria-laden protein coagulum within a capacious upper fornix. Persistent reinoculation of the tear lake by low-grade bacteria from the fornix, severe conjunctivitis from 'toxic' bacteria and the development of a pseudo-membrane, may enhance the coagulum and also exacerbate the ptosis and deepening of the upper fornix. This environment leads to severe ocular surface inflammation.

This case highlights the need to consider a deep upper fornix, present in many of our patients with age-related levator disinsertion and orbital fat atrophy, as the source of a recurrent (often unilateral) purulent discharge. In addition, we suggest that CT imaging is beneficial in excluding frontal sinus fistulae and foreign bodies in such cases, and the presence of air in the deep upper fornix of this patient on CT scan is a new sign of GFS, not described previously.

Reference

1 Rose GE. The giant fornix syndrome: an unrecognised cause of chronic, relapsing, and grossly purulent conjunctivitis. *Ophthalmology* 2004; **111**: 1539–1545.

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Sir,

Third nerve paralysis as a presenting sign of essential thrombocythaemia

Essential thrombocythaemia is a clonal myeloproliferative disease characterised by a sustained platelet count in excess of 600×10^9/L and clinically, by episodes of thrombosis and/or haemorrhage.[1] The thrombotic events primarily affect the microvasculature. Ocular complications are rare and include central retinal vein and central retinal artery occlusion. We report the clinical findings in a patient with essential thrombocythaemia who presented with partial third nerve paralysis; an association not previously described.

Case report

A 74-year-old man presented with a 1-day history of double vision associated with a right-sided frontal headache. He was systemically well and not on any regular medications. He was not a smoker. Both temporal arteries were pulsatile and nontender and the patient had no other symptoms of giant cell arteritis. On ophthalmological examination, the visual acuity was 6/6 in each eye and colour vision was normal. The ocular examination including the pupils and optic discs was entirely normal in both eyes. Evaluation of the extraocular movements showed limitation of adduction, elevation, and infraduction in the right eye. No ptosis was detected. Examination of the other cranial nerves was within normal limits. These findings were consistent with a diagnosis of pupil-sparing partial third nerve paralysis.

At presentation, the patient's blood pressure was raised at 160/100 mmHg. MRI scan of the orbits and brain was completely normal. The ESR, CRP, blood lipid, and blood glucose levels were within normal limits. Further haematologic studies showed neutrophilia (18×10^9/l) and an extremely high platelet count of 1024×10^9/l (normal $150–450 \times 10^9$/L). Bone marrow biopsy was performed, which revealed a hypercellular marrow with atypical megakaryocytes but no marrow fibrosis was noted. Chromosomal analysis was normal thus excluding a diagnosis of chronic myeloid leukaemia. Essential thrombocythaemia was diagnosed and the patient was started on hydroxycarbamide and aspirin. The blood pressure was controlled with medication. Within 2 weeks the platelet count decreased to 315×10^9/l. The diplopia improved rapidly and the patient was essentially symptom free a month after starting treatment. At the end of 5 months of follow-up, his platelet counts remained stable on medication and the third nerve paralysis had almost completely resolved.

Comment

To our knowledge, this is the first report of an association between essential thrombocythaemia and third nerve

paralysis. Essential thrombocythaemia is a rare condition with an incidence of around 1–2.5 per 100 000 per year. It affects both sexes and is most common in the sixth and seventh decades. More than one-half of the patients are asymptomatic when the condition is detected on a routine full blood count.[1] Untreated, patients are at increased risk of cerebrovascular accidents, deep vein thrombosis, and gastrointestinal tract bleeding. Neurologic symptoms are common and include headache, paresthesiae, and amaurosis fugax.[2] A small number of case reports of central retinal vein occlusion and central retinal artery occlusion in essential thrombocythaemia have been published.[3–6]

The differential diagnosis of a persistently high platelet count includes polycythaemia vera (increased red cell mass in the presence of normal iron stores), chronic myeloid leukaemia (presence of the Philadelphia chromosome) and agnogenic myeloid metaplasia (prominent marrow fibrosis).

Essential thrombocythaemia is known to affect the microvasculature and we hypothesise that the thrombocytosis contributed to the ischaemic third nerve palsy in our patient. Although this patient had increased blood pressure at presentation, two large studies have shown that hypertension is not an independent risk factor for ocular motor nerve palsies.[7,8] While we accept that hypertension may have played a contributory role, we believe that the presence of headache (a common symptom in patients with essential thrombocythaemia) and the rapid resolution of symptoms following initiation of treatment support our hypothesis that essential thrombocythaemia was a precipitating, if not sole causative factor, for the third nerve palsy in this case.

Reports of third nerve palsy secondary to other haematologic abnormalities are also very uncommon. However, it is interesting to note that, in the two reported cases of third nerve palsy secondary to monoclonal gammopathy,[9,10] pain was a presenting feature. This may suggest involvement of the third nerve in the cavernous sinus as sensory fibres from the ophthalmic division of the fifth nerve join the third nerve in the lateral wall of the cavernous sinus. Essential thrombocythaemia should be included in the differential diagnosis of ischaemic ocular motor nerve palsies. This case also illustrates the importance of routine haematology work-up in cases of spontaneous ocular motor nerve palsies.

References

1 Imbert M, Pierre R, Thiele J, Vardiman JW, Brunning RD, Flandrin G. Essential thrombocythaemia. In: Jaffe ES, Harris NL, Stein H, Vardiman JW (eds) *WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues*. IARC Press:Lyon, 2001 pp 39–41.
2 Jabaily J, Iland HJ, Laszlo J, Massey EW, Faguet GB, Briere J *et al*. Neurological manifestations of essential thrombocythemia. *Ann Intern Med* 1983; **99**: 513–518.
3 Yoshizumi MO, Townsend-Pico W. Essential thrombocythemia and central retinal vein occlusion with neovascular glaucoma. *Am J Ophthalmol* 1996; **121**(6): 728–730.
4 Imasawa M, Iijima H. Multiple retinal vein occlusions in essential thrombocythemia. *Am J Ophthalmol* 2002; **133**(1): 152–155.
5 Liu M, Lee AG, Rice L, Lambert HM. Bilateral retinal vascular occlusive disease in essential thrombocythemia. *Retina* 1999; **19**(6): 563–564.
6 Strassman I, Silverstone BZ, Seelenfreund MH, Sheer A, Berson D. Essential thrombocythemia: a rare cause of central retinal artery occlusion. *Metabol Pediatr Syst Ophthalmol* 1991; **14**: 18–20.
7 Patel SV, Holmes JM, Hodge DO, Burke JP. Diabetes and hypertension in isolated sixth nerve palsy: a population-based study. *Ophthalmology* 2005; **112**(5): 760–763.
8 Jacobson DM, McCanna TD, Layde PM. Risk factors for ischemic ocular motor nerve palsies. *Arch Ophthalmol* 1994; **112**: 961–966.
9 Bogousslavsky J, Steck AJ. Bilateral third nerve palsy and anterior ischemic optic neuropathy. *Neuro-ophthalmology* 1986; **6**: 117–120.
10 Ruggles KH, Massey EW. Pupil-sparing oculomotor palsy with IgG momoclonal paraproteinemia. *Ann Ophthalmol* 1981; **13**: 875–876.

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Sir,

Intrableb triamcinolone acetonide injection after bleb-forming filtration surgery (trabeculectomy, phacotrabeculectomy, and trabeculectomy revision by needling): a pilot study

Bleb-forming filtration procedures, such as trabeculectomy, combined phacotrabeculectomy, and trabeculectomy revision by needling, are surgical options in medically uncontrolled glaucoma. Postoperative topical steroid significantly increased the chance of